

# Prenatal exposure to passive smoking and duration of breastfeeding in nonsmoking women: Krakow inner city prospective cohort study

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## Abstract

**Introduction** The relationship between tobacco smoking in pregnancy and breastfeeding is of public health importance. The present birth cohort study provided the opportunity to investigate whether the negative relationship between passive smoking measured by the cotinine concentrations in maternal blood at delivery and breastfeeding in postpartum could also be confirmed in nonsmoking mothers.

**Materials and methods** The study sample included 441 healthy pregnant women who were recruited in the first and second trimester of pregnancy. Enrolment included only nonsmoking women of the age of 18–35 years with singleton pregnancies, without illicit drug use and free from chronic diseases. After delivery, breastfeeding duration was defined using the answers recorded in the interviews with mothers conducted every 3 months. An infant was considered to be fully breastfed when breast milk was the only source of nourishment. Any breastfeeding was defined as an infant's being fully breastfed or receiving both breast milk and formula, with or without solids. In the statistical analysis only total duration of breastfeeding up to 6 months was considered for both forms of breastfeeding. Subjects were categorized into environmental tobacco smoke (ETS) exposure groups according to maternal blood cotinine level at delivery or self-reported exposure to ETS during pregnancy.

**Results** The adjusted relative risk of discontinuation of any breastfeeding after infant's first 6 months was more than two times higher (OR = 2.42; 95% CI: 1.42–4.14) in women whose blood cotinine level at delivery was above 75th percentile of cotinine distribution (>0.15 ng/mL); the corresponding risk of discontinuation of full breastfeeding was OR = 1.71; 95% CI: 1.03–2.82. Estimated relative risk of discontinuation of any breastfeeding based on self-reported ETS was also significant but much less marked; the corresponding risk of discontinuation of full breastfeeding was insignificant.

**Conclusion** The results obtained support the hypothesis that ETS may affect breastfeeding duration and support the avoidance of passive smoking as a necessary additional measure for breastfeeding promotion.

**Keywords** Prenatal ETS exposure · Maternal cotinine · Breastfeeding duration

## Introduction

A report of an adverse effect of maternal smoking in pregnancy on breastfeeding was first published by Mills in 1950 [1], and since then many other authors described the negative effect of smoking on breastfeeding duration [2–13]. In the meta-analysis of 13 studies Horta et al. [14] compared breastfeeding duration in smoking and nonsmoking mothers, where the odds ratio for weaning before 3 months was 1.94 (95% CI: 1.55–2.40). An adjusted ratio of 1.50 (95% CI: 1.34–1.68) was shown in the studies, where the rate of lost-to-follow-up was below 15% and the adjustment for confounding was adequate. Although some studies did not confirm the adverse effects of smoking on breastfeeding [15], it is now generally believed that maternal smoking has

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a detrimental effect on breast milk supply and that women who smoke are less likely to breastfeed their children.

The causal association between smoking and breastfeeding may be confounded by the lower motivation of smoking women to breastfeed and less likely to initiate breastfeeding, as was shown in the recent ALSPAC study and reported by other authors [16–21]. The independent effect of smoking was to increase the risk of discontinuation of breastfeeding at any time by 17%. However, the independent effect of maternal breastfeeding intention was greater than the effect of smoking, with intention to breastfeed for 2–4 months increasing the risk of stopping breastfeeding by 51% (compared with those intending to breastfeed for at least 4 months), and intention to breastfeed for only 1 month increasing the risk by 78%.

Unfortunately, many published studies have neither properly accounted for the socio-demographic differences between women who smoke and nonsmokers, nor considered important confounders. Moreover, in a majority of studies on pregnancy and lactation, smoking was measured by maternal self-reports. Since self-reported use of cigarettes tends to be underestimated the observed effect of smoking on breastfeeding duration may be biased.

In the present birth cohort study in Krakow we investigated whether the negative relationship between environmental tobacco smoke (ETS) measured by the cotinine concentrations in maternal blood at delivery and breastfeeding in postpartum could also be confirmed in never smoking mothers. Cotinine, being a major metabolite of nicotine and having a much longer half-life than nicotine, is used as a most specific and sensitive biomarker of cigarette-smoke exposure in epidemiological studies. Recent advances in laboratory methods allow precise evaluation of cotinine levels even in the blood of passive smokers. Moreover, our study design based only on nonsmoking women would exclude the importance of maternal active smoking as a variable possibly associated with a complex constellation of factors strongly correlated with social and environmental influences having potential impact on breastfeeding practices.

## Materials and methods

This study uses data from an earlier established Krakow birth cohort of children being the part of the collaborative study with Columbia University in New York. The design of the study and the detailed selection of the population have been described previously [22]. Briefly, between November 2001 and February 2004, a total of 505 women recruited from ambulatory prenatal clinics and healthy pregnant women in the first and second trimester of pregnancy delivered babies. Prior to participation, women read

and signed an informed consent. The Ethical Committee of the Jagiellonian University approved the research.

The enrolment included only women 18–35 years of age, who claimed to be nonsmokers, with singleton pregnancies, without illicit drug use and HIV infection, free from chronic diseases such as diabetes or hypertension, and residents of Krakow for at least 1 year prior to pregnancy. Upon enrolment, a detailed questionnaire was administered to each woman at the entry to the study to solicit information on demographic data, house characteristics, medical and reproductive history, occupational hazards, and smoking practices of others present in the home. The questionnaire on passive smoking included questions on how many members of the household smoked cigarettes and how many cigarettes were smoked daily in front of a given respondent in the second and third trimester of pregnancy. The latter information was used to calculate the weighted average number of cigarettes smoked daily in the presence of pregnant woman.

After delivery, breastfeeding duration was defined using the answers recorded at each interview with mother conducted every 3 months. Mothers were asked whether the infant had ever been breastfed and whether infant received other liquids or solids. An infant was considered to be fully breastfed when breast milk was the only source of nourishment. Any breastfeeding was defined as an infant's being fully breastfed or receiving both breast milk and formula, with or without solids. In the analysis, only total duration of breastfeeding up to 6 months was considered for both forms of breastfeeding. The cut-off for breastfeeding duration results from the fact that it is widely recommended that women should breastfeed their infants for the first 6 months.

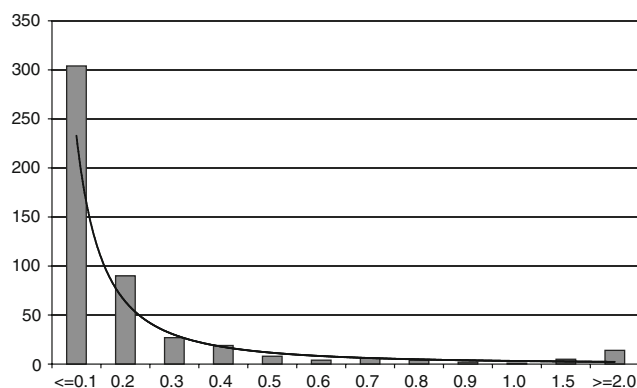
Women at delivery provided a blood specimen and the blood samples before laboratory analysis were stored at  $-70^{\circ}\text{C}$ . The serum cotinine concentration was measured at CDC using a sensitive isotope-dilution high-performance liquid chromatographic/atmospheric pressure ionization tandem spectrometric (LC/MS/MS) procedure [23]. Limits of detection (LOD) were below 0.05 ng/mL. About 25% of specimens had cotinine levels below the LOD. Maternal blood cotinine level below 15.0 ng/mL was considered the borderline separating smokers from nonsmokers [24, 25].

The main purpose of the statistical analysis was to correlate the prenatal passive smoking measured by the maternal cotinine level at delivery and self-reported ETS exposure with the outcome variables (duration of breastfeeding in postpartum period) over the first 6 months in postpartum period. Differences between subgroups by ETS exposure were tested by Chi-square statistics (categorical variables) or by *t*-test (numerical variables). Logistic multiple regression models were used to analyze the association between ETS and the postpartum breastfeeding duration (number of

weeks) recorded over the follow up. In the models, a set of potential confounders such as maternal education, parity, gender of child, gestational age, and maternal prepregnancy body mass index (BMI) were included as independent variables. In the multivariate analysis two models have been used. In the first model the cotinine level was divided into three categories (1)  $<0.05$  ng/mL; (2)  $0.05$ – $0.15$  ng/mL, and (3)  $>0.15$  ng/mL (75th percentile of the cotinine distribution in the total study sample). In the second model the self-reported ETS exposure (number of cigarettes smoked daily by other members of the household) was introduced. Out of the total recruited women the cotinine measurements were done in 467 persons but in the final multivariate statistical analysis the data from 441 women with the completed database were included. Out of 64 women excluded from the analysis 17 showed blood cotinine level above  $15$  ng/mL, which could indicate active smoking in pregnancy, 5 women lacked cotinine measurements, 3 reported smoking in postpartum period and 39 persons were lost to follow-up. Statistical analyses were performed with STATA 10 software for Windows [26, 27].

## Results

Out of the total study sample 332 (75.3%) respondents denied exposure to passive smoking, 97 (22.0%) were exposed to ten or less cigarettes smoked daily at home by other household members, and only 12 (2.7%) reported the ETS exposure to more than ten cigarettes smoked daily. Distribution of blood cotinine concentrations was very skew (Fig. 1). Median concentration of maternal blood cotinine obtained from the total study sample was  $0.072$  ng/mL (95% CI:  $0.067$ – $0.08$ ). Spearman's coefficient of rank correlation between reported average number of cigarettes smoked daily at home and maternal blood cotinine measured at delivery was significant ( $r = 0.54$ ,  $p < 0.0001$ ).



**Fig. 1** Distribution of maternal blood cotinine levels (ng/mL)

In total, 99% of women who delivered babies initiated breastfeeding in the first week after delivery. At infant's 6 months of age, about one half (48.4%) of infants were fully breastfed and 79.1% received any breast milk. Women who were exposed to higher cotinine level ( $>0.15$  ng/mL) were younger, less educated and reported shorter breastfeeding than those with lower exposure. It is worthwhile to notice that returning to work after pregnancy did not differ across the ETS exposure groups (Table 1).

Spearman crude rank correlation coefficients between maternal blood cotinine levels and duration of any breastfeeding (weeks) ( $r = -0.198$ ,  $p < 0.0001$ ) and for the full breastfeeding ( $r = -0.195$ ,  $p < 0.0001$ ) were statistically significant. Rank correlation between any breastfeeding and self-reported passive smoking was  $r = -0.187$  ( $p = 0.0001$ ) and that for full breastfeeding  $r = -0.155$  ( $p = 0.0011$ ).

Predicted probability of any breastfeeding discontinuation before 6 months—estimated from the multiple logistic model and adjusted to maternal age, overweight, parity, gestational age, and gender of child—correlated significantly with the maternal cotinine concentrations (Spearman rank correlation coefficient =  $0.29$  (95% CI:  $0.20$ – $0.37$ ,  $p = 0.0001$ ), and suggested a linear dose–response relationship between the prenatal ETS exposure and breastfeeding (Fig. 2).

The adjusted relative risk (OR) for stopping any breastfeeding at 6 months was more than two times higher (OR =  $2.42$ ; 95% CI:  $1.42$ – $4.14$ ) in women whose blood cotinine level at delivery was above 75th percentile of the distribution ( $>0.15$  ng/mL) (Table 2), compared with mothers who had lower cotinine level; the corresponding risk for discontinuation of full breastfeeding was OR =  $1.71$ ; 95% CI:  $1.03$ – $2.82$  (Table 3). Estimate of adjusted relative risk for discontinuation of any breastfeeding related to self-reported passive smoking was also significantly higher (OR =  $1.07$ ; 95% CI:  $1.01$ – $1.13$ ) (Table 4), while the corresponding risk of discontinuation of full breastfeeding did not show statistical significance (Table 5).

The risk of discontinuation of any breastfeeding before 6 months postpartum increased more than twofold in more exposed mothers. The risk of full breastfeeding discontinuation at any time before 6 months due to higher exposure was 71% higher compared with mothers who had lower level of blood cotinine. The results of the analysis with self-reported ETS suggest that the risk of giving up any breastfeeding 6 months postpartum increased by 70% for reported ten cigarettes smoked daily in the household. Interestingly, maternal education level was consistently inversely related to both types of breastfeeding practices. The breastfeeding of the first child lasted also significantly shorter, and prepregnancy body mass index significantly increased the risk of discontinuation of any breastfeeding before 6 months postpartum.

**Table 1** Characteristics of the study subjects by the cotinine exposure level

Variables (maternal characteristics)	Total	Cotinine (maternal blood) exposure level		
		<0.15 ng/mL (N = 346)	>0.15 ng/mL (N = 121)	<i>p</i>
Mother's age				
Mean	27.74	28.11	26.68	0.0001
SD	3.48	3.31	3.75	
Education				
Elementary	39 (8.4%)	20 (5.8%)	19 (15.7%)	0.0001
Intermediary	112 (24.0%)	74 (21.4%)	38 (31.4%)	
Higher and university	316 (67.7%)	252 (72.8%)	64 (52.9%)	
Mother's height (cm)				
Mean	165.06	165.07	165.04	0.9652
SD	5.44	5.18	6.16	
Mother's weight (kg) (before pregnancy)				
Mean	58.17	58.09	58.41	0.7266
SD	8.45	8.19	9.18	
Overweight (before pregnancy) <i>n</i> (%)	40 (8.6%)	28 (8.1%)	12 (9.9%)	0.6743
Parity <sup>a</sup>				
1	294 (63.0%)	218 (63.0%)	76 (62.8%)	1.0000
32	173 (37.0%)	128 (37.0%)	45 (37.2%)	
Any breastfeeding (weeks)				
Mean	45.32	48.75	35.50	0.0001
SD	31.56	30.99	31.27	
Median	40.00	44.50	27.00	
Q <sub>3</sub> –Q <sub>1</sub> /2	21.50	19.50	23.50	
Full breastfeeding (weeks)				
Mean	26.86	28.84	21.18	0.0008
SD	21.69	21.89	20.14	
Median	19.00	32.00	19.00	
Q <sub>3</sub> –Q <sub>1</sub> /2	17.50	17.50	13.00	
Return to work after delivery				
≤6 months	193 (44.3%)	138 (43.1%)	55 (50.0%)	0.3640
7–18 months	82 (19.3%)	67 (20.9%)	15 (13.6%)	
19 months or later	114 (26.3%)	85 (26.6%)	29 (26.4%)	
Missing data	41 (10.1%)	30 (9.4%)	11 (10.0%)	
	37	26	11	

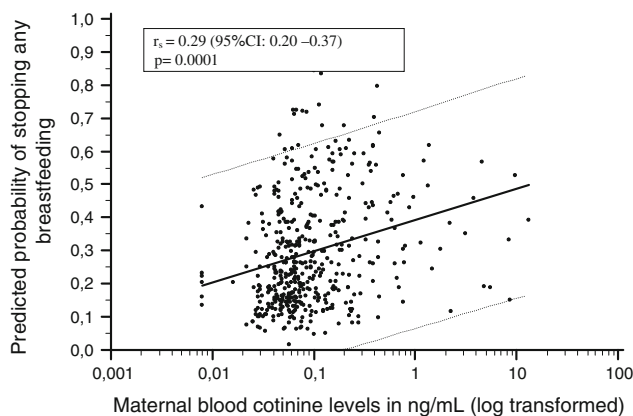
<sup>a</sup> values are *n* (%)

## Discussion

The study has shown that duration of full or any breastfeeding in postpartum correlated inversely with prenatal ETS exposure measured by maternal blood cotinine level at delivery or by self-reported exposure to passive smoking in pregnancy. The risk for stopping any breastfeeding before an infant's first 6 months more than doubled in women with higher cotinine levels compared with those who displayed lower levels. The risk of stopping full breastfeeding over the first 6 months was 71% higher in mothers who had maternal cotinine level above 0.15 ng/mL. The comparative analysis using the self-reported ETS exposure (average

number of cigarettes smoked daily by household members) instead of the blood cotinine measurements showed a weaker association with breastfeeding duration. Interestingly, maternal education level was consistently inversely related to both types of breastfeeding practices, however prepregnancy body mass index increased the risk of discontinuing any breastfeeding in the first 6 months postpartum.

The results of our study are consistent with many authors who believe that smoking mothers are more likely to give up breastfeeding than other women [2–13]. There are a number of possible explanations for an adverse effect of cigarette smoke on breastfeeding. These include a physiological effect of nicotine on mother's hormonal system or



**Fig. 2** Probability of stopping any breastfeeding before 24 months and maternal blood cotinine levels (log transformed)

**Table 2** Multiple logistic regression for the risk for discontinuing any breastfeeding before 6 months

Predictors	Odds ratio	Std. err.	z	p > z	95% CI
Maternal education <sup>a</sup>	0.071	0.058	−4.23	0.000	0.601 0.830
Parity <sup>b</sup>	0.729	0.138	−1.67	0.095	0.502 1.057
Gender of child <sup>c</sup>	0.666	0.151	−1.79	0.074	0.427 1.040
Gestational age (weeks)	0.821	0.058	−2.78	0.005	0.714 0.943
Cotinine level (0.05–0.15 ng/mL)	1.079	0.311	0.26	0.792	0.614 1.898
Cotinine level (>0.15 ng/mL)	2.424	0.662	3.24	0.001	1.419 4.141
Overweight <sup>d</sup>	3.429	1.338	3.16	0.002	1.596 7.366

ETS exposure in pregnancy expressed by the maternal cotinine level

<sup>a</sup> Maternal education: 1 elementary, 2 intermediary, 3 higher and university

<sup>b</sup> Parity: 1 first child, 2 other

<sup>c</sup> Gender of child: 0 boys, 1 girls

<sup>d</sup> Overweight: 0 BMI ≤ 25, 1 BMI > 25

on her breasts directly [28–34]. The mechanism is generally thought to be lowering of prolactin by nicotine [28]. As nicotine leads to vasoconstriction, a decreased blood flow to the breast could reduce levels of circulating oxytocin, which would reduce the milk available to the infant [18]. Despite this evidence, we should not forget that smoking may still be acting as a proxy for behavioural factors, which may play more important role in lowering rates of breastfeeding found in smokers, than any physiological effect of smoking [6, 19–21].

The consistency of our findings with previous studies on maternal smoking in pregnancy and breastfeeding makes more likely the hypothesis of a potential causal association between ETS and breastfeeding duration. Since ETS operates through the mother, it may influence breastfeeding by

**Table 3** Logistic multiple regression for the risk for discontinuing full breastfeeding before 6 months

Predictors	Odds ratio	Std. err.	z	p > z	95% CI
Maternal education <sup>a</sup>	0.808	0.062	−2.80	0.005	0.695 0.938
Parity <sup>b</sup>	0.671	0.110	−2.44	0.015	0.487 0.925
Gender of child <sup>c</sup>	0.648	0.129	−2.18	0.029	0.439 0.957
Gestational age (weeks)	0.950	0.061	−0.80	0.425	0.837 1.078
Cotinine level (0.05–0.15 ng/mL)	1.192	0.294	0.71	0.478	0.735 1.932
Cotinine level (>0.15 ng/mL)	1.706	0.437	2.08	0.037	1.033 2.819
Overweight <sup>d</sup>	1.733	0.655	1.45	0.146	0.826 3.633

ETS exposure in pregnancy expressed by the maternal cotinine level

<sup>a</sup> Maternal education: 1 elementary, 2 intermediary, 3 higher and university

<sup>b</sup> Parity: 1 first child, 2 other

<sup>c</sup> Gender of child: 0 boys, 1 girls

<sup>d</sup> Overweight: 0 BMI ≤ 25, 1 BMI > 25

**Table 4** Logistic multiple regression for the risk for discontinuing any breastfeeding before 6 months

Predictors	Odds ratio	Std. err.	z	p > z	95% CI
Maternal education <sup>a</sup>	0.700	0.057	−4.38	0.000	0.597 0.821
Parity <sup>b</sup>	0.755	0.141	−1.51	0.131	0.524 1.088
Gender of child <sup>c</sup>	0.627	0.141	−2.08	0.038	0.404 0.974
Gestational age (weeks)	0.802	0.057	−3.11	0.002	0.698 0.922
Self-reported ETS	1.067	0.033	2.11	0.035	1.005 1.133
Overweight <sup>d</sup>	3.265	1.256	3.08	0.002	1.537 6.938

ETS exposure in pregnancy expressed by the self-reported ETS exposure

<sup>a</sup> Maternal education: 1 elementary, 2 intermediary, 3 higher and university

<sup>b</sup> Parity: 1 first child, 2 other

<sup>c</sup> Gender of child: 0 boys, 1 girls

<sup>d</sup> Overweight: 0 BMI ≤ 25, 1 BMI > 25

an effect on prolactin production. Eventually, ETS exposure may perhaps change the taste of human milk, making it less tasty to children. Moreover, ETS increasing the frequency of recurrent respiratory infections in offspring [35–41] can affect the child's desire for food and weaken nipple stimulation and reduce milk production. It is worth mentioning that our effect estimates of passive smoking on breastfeeding would be close enough in magnitude to effects attributed to active maternal smoking in pregnancy [14].

The ETS effect on breastfeeding has first been mentioned in the literature by Horta et al. [42], however, their findings were not supported by cotinine measurements. Compared



**Table 5** Logistic multiple regression for the risk for discontinuing full breastfeeding before 6 months

Predictors	Odds ratio	Std. err.	z	P > z	95% CI
Maternal education <sup>a</sup>	0.793	0.060	−3.05	0.002	0.683 0.920
Parity <sup>b</sup>	0.671	0.108	−2.48	0.013	0.489 0.920
Gender of child <sup>c</sup>	0.638	0.126	−2.28	0.023	0.434 0.940
Gestational age (weeks)	0.935	0.060	−1.04	0.298	0.825 1.061
Self-reported ETS	1.035	0.033	1.09	0.275	0.973 1.101
Overweight <sup>d</sup>	1.693	0.638	1.40	0.162	0.809 3.541

ETS exposure in pregnancy expressed by the self-reported ETS exposure

<sup>a</sup> Maternal education: 1 elementary, 2 intermediary, 3 higher and university

<sup>b</sup> Parity: 1 first child, 2 other

<sup>c</sup> Gender of child: 0 boys, 1 girls

<sup>d</sup> Overweight: 0 BMI ≤ 25, 1 BMI > 25

with nonsmokers, mothers smoking 20 or more cigarettes daily presented an odds ratio of 1.94 for any breastfeeding for less than 6 months. Environmental tobacco smoke was also an independent risk factor. After adjustment for maternal smoking and other confounders, households where more than ten cigarettes were smoked daily by persons other than the mother presented odds ratio of 1.48 compared with those without smokers. These results remained unchanged after stratification for maternal smoking.

The documented, by us, potential hazard of ETS related to breastfeeding duration expand our knowledge on already well-documented negative impact of passive smoking on child health, including that on low birth weight and respiratory infections. The strength of our evidence comes from the fact that our study concerning only nonsmoking women excluded maternal active smoking as a variable possibly associated with a complex constellation of factors strongly correlated with social and environmental influences having potential impact on breastfeeding practices. The strength of our study lies not only in the large population-based sample and in measuring ETS exposure by maternal blood cotinine level, but also in our adjustment for a set of socio-demographic confounders, which were not considered in earlier studies. For example, prepregnancy body mass index, which was shown to be consistent and inversely related to breastfeeding duration. An importance of prepregnancy body mass has earlier been documented [43, 44]. Rasmussen and Kjolhede [44] have recently shown that initiation and duration of breastfeeding, is negatively affected by maternal overweight/obesity and that overweight/obese women had a lower prolactin response to suckling. This could compromise the ability of overweight/obese women to produce milk and subsequently lead to premature cessa-

tion of lactation. The authors postulated that consistently higher progesterone concentrations in the early postpartum period among obese compared with normal-weight women would have an adverse impact on breastfeeding since adipose tissue is an extraplacental source of this hormone.

Taken together, our results strongly suggest that environmental tobacco smoking affects breastfeeding duration. The data support the avoidance of passive smoking as a necessary additional measure in breastfeeding promotion. Although generally, women have the knowledge that smoking is harmful, they may not be aware of the harmful effect of ETS. Therefore, the message about the hazards of prenatal environmental tobacco smoke should be provided to prospective potential mothers and the public at large.

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